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Meta-Analysis

Corticosteroids for community-acquired pneumonia in non-ICU patients presenting to the emergency department: a systematic review and meta-analysis

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ABSTRACT

Community-acquired pneumonia (CAP) is among the most common infectious indications for emergency department attendance and hospitalization and carries a heavy mortality burden despite advances in antimicrobial therapy. Because a dysregulated host inflammatory response independently drives clinical deterioration, corticosteroids have been proposed as adjunctive treatment, but their benefit in non-ICU patients presenting to the emergency department remains unclear. A systematic search of PubMed, Web of Science, and EMBASE was conducted to identify randomized controlled trials evaluating systemic corticosteroids versus placebo or standard care in adults with CAP managed outside the ICU. The primary outcome was all-cause mortality; secondary outcomes included time to clinical stability, length of hospital stays, ICU admission, and adverse events. Twelve RCTs were included. Pooled analysis showed no significant difference in mortality between the corticosteroid and control groups (RR=0.98, 95% CI: 0.87–1.10, p=0.71; I^2=0%). Neither time to clinical stability nor length of hospital stay differed significantly between groups, though both outcomes showed substantial heterogeneity. Corticosteroid use was associated with a significantly higher risk of hospital readmission (RR=1.35, 95% CI: 1.11–1.64, p<0.001) and hyperglycemia (RR=1.69, 95% CI: 1.49–1.92, p<0.001). Early corticosteroid therapy in non-ICU adults with CAP does not reduce mortality or meaningfully accelerate clinical recovery, while consistently increasing the risks of hyperglycemia and hospital readmission. Routine use in this population cannot be recommended on current evidence, and future trials should incorporate biomarker-guided designs to identify inflammatory phenotypes most likely to benefit from corticosteroid therapy.

Keywords: Community-acquired pneumonia, Corticosteroids, Emergency department, Systematic review and meta-analysis, Clinical outcomes

INTRODUCTION

Community-acquired pneumonia (CAP) is one of the most common and consequential infections encountered in acute care settings, ranking among the leading infectious causes of hospitalization, emergency department (ED) attendances, and death in both high- and low-income countries.[1] Well, With advances in antibiotics and supportive care, an influential portion of patients' experience protracted clinical courses characterized by delayed retrieval, prolonged inpatient stays, and occasionally treatment failure. This persistent burden raises an important and unresolved question: whether the pathophysiological response to infection itself drives poor consequences and whether it can be therapeutically changed.

The inflammatory milieu in CAP is widely acknowledged as a double-edged phenomenon. While an immune response is essential for pathogen clearance, an exaggerated or dysregulated cascade of cytokine release and alveolar injury appears to contribute unassisted to clinical deterioration.[2] This biological rationale has protracted underpinned curiosity in systemic corticosteroids as an adjunctive method, given their capability to attenuate inflammatory signaling without directly targeting the organism.[3]

Early trials provided grounds for cautious optimism, with some reporting shorter time to clinical stabilization and reduced hospital stay; yet the cumulative evidence on mortality has remained frustratingly mixed, and concerns about harm particularly hyperglycemia, immunosuppression, and secondary infections have tempered enthusiasm.[4]

The emergency department occupies a uniquely pivotal moment in the CAP illness trajectory. Decisions made at the point of initial presentation including whether to initiate adjunctive anti-inflammatory therapy may set the clinical course before disease severity fully declares itself. Yet this is precisely the setting and patient population least well characterized in the existing literature.

Most landmark trials have enrolled either broadly defined hospitalized cohorts or patients already triaged to the ICU, leaving non-ICU patients managed through or beyond the ED in a relative evidence vacuum. Current clinical guidelines reflect this ambiguity, offering divergent or insufficiently qualified recommendations.[4]

The aim of this systematic review and meta-analysis is to evaluate the effectiveness and safety of early systemic corticosteroid therapy, initiated at emergency department presentation or within 24 hours of arrival, compared with standard care or placebo, in adults with community-acquired pneumonia who do not require intensive care unit admission, with respect to mortality, clinical recovery, disease deterioration, and adverse events.

METHODS

Study protocol and registration

This study was conducted and reported in accordance with the preferred reporting items for systematic reviews and meta-analyses (PRISMA) 2020 framework.[5] The protocol was registered a priori in PROSPERO (CRD420261287797).

Eligibility criteria

Inclusion was restricted to randomized controlled trials or quasi-randomized controlled trials that enrolled adults aged 18 years or older presenting to emergency departments with radiographically confirmed community-acquired pneumonia. Eligible studies included patients managed outside intensive care units at initial presentation who received systemic corticosteroids either during emergency department evaluation or within 24 hours of arrival. Comparator groups were required to receive either a placebo or standard care protocols that strictly excluded the use of corticosteroids. Studies were also required to report at least one clinical outcome including mortality, time to clinical stability, length of hospital stay, ICU admission, or adverse events with a minimum follow-up of seven days or until hospital discharge.

Exclusion criteria included studies involving pediatric populations, hospital-acquired or ventilator-associated pneumonia, or patients requiring intensive care units' admission at baseline presentation. Studies focusing exclusively on immunocompromised populations were also excluded. Additional exclusions were applied to studies involving chronic systemic corticosteroid use prior to presentation, use of inhaled corticosteroids alone, or corticosteroid administration for indications other than pneumonia. Additionally, retrospective studies, case reports, case series, review articles, editorials, conference abstracts without full-text availability, non-randomized studies, studies lacking extractable outcome data, non-English language publications, and animal studies models were excluded.

Search strategy and study selection

A comprehensive systematic search was executed across three electronic databases: PubMed, Web of Science, and EMBASE, using predefined search terms. The search strategy employed the following Boolean combinations: ("community-acquired pneumonia" OR pneumonia) AND (corticosteroids OR systemic steroids OR prednisone OR methylprednisolone OR hydrocortisone) AND (emergency department OR non-ICU) AND (randomized controlled trial). No temporal restrictions were applied to ensure comprehensive identification of relevant studies.

Database searches were performed by a single reviewer. The study selection process consisted of three phases. Initially, three independent reviewers screened titles and

abstracts using the Rayyan platform (QCRI), with blinding enabled to maintain independence. Second, four independent reviewers assessed the full texts of potentially eligible studies to evaluate compliance with established inclusion criteria. Discrepancies at either stage were reconciled through consensus deliberation between two reviewers

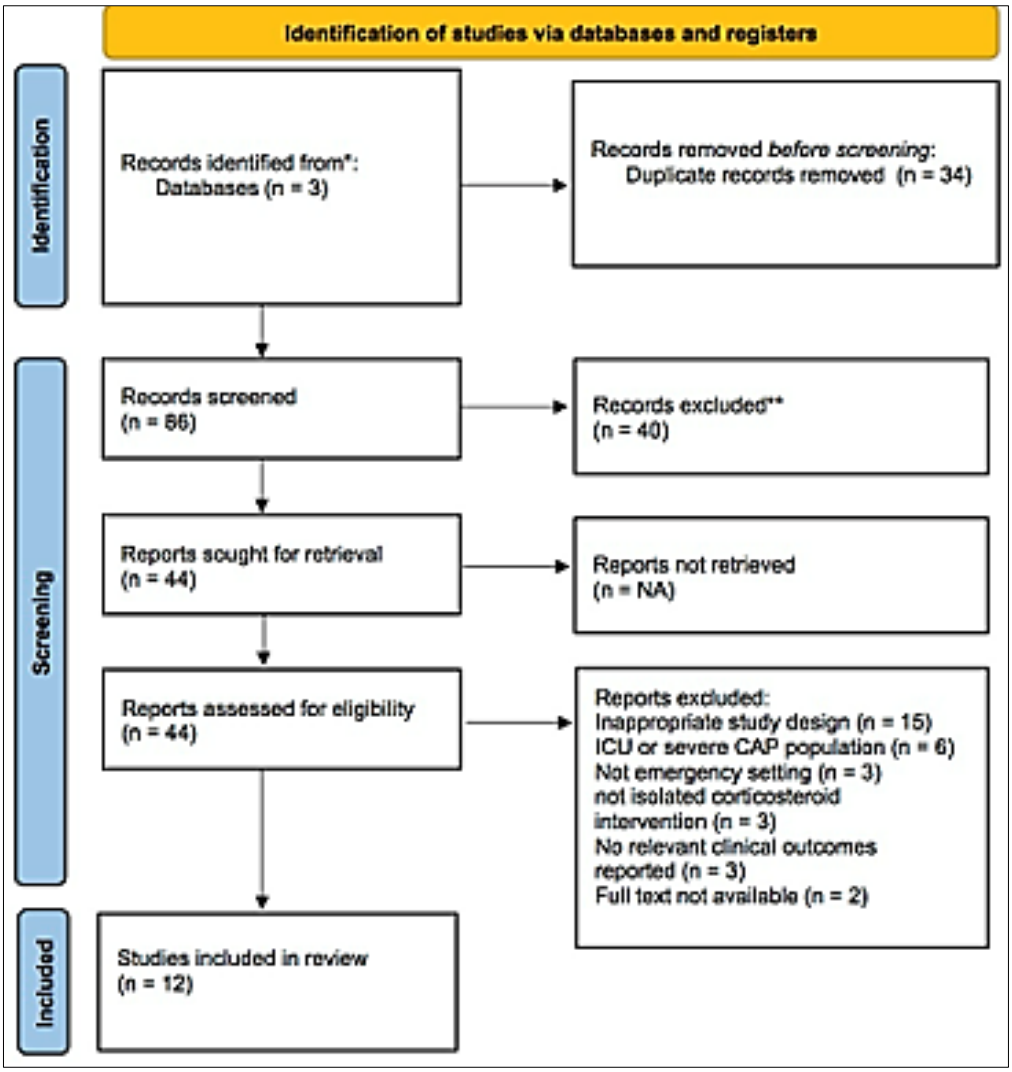

Figure 1: PRISMA 2020 flow diagram.

The following PRISMA 2020 flow diagram delineates the study selection process, spanning from initial database identification to final inclusion. Records were retrieved from PubMed, Web of Science, and EMBASE. Following the elimination of duplicates, titles and abstracts were screened independently by three reviewers. Eligible full-text articles were assessed against the established inclusion and exclusion criteria. Twelve randomized controlled trials ultimately met all eligibility criteria and were incorporated in the systematic review and meta-analysis.

Data extraction

Data extraction was performed independently by four reviewers using a standardized data extraction form. Extracted data included study characteristics (author, year of publication, country, study design, and sample size) and participant characteristics (age, sex, baseline disease severity, and comorbidities). Intervention details were also documented, including corticosteroid type, dosage, route of administration, timing of initiation, and duration of therapy. Comparator details including components of standard care and placebo were documented. Outcome measures extracted included mortality, time to clinical stabilization, length of hospital stay, ICU admission, and reported adverse events. Discrepancies were resolved through discussion and consensus or, when necessary, by consultation with the lead author.

Risk of bias assessment

The methodological quality of the included studies was systematically evaluated using the Cochrane Risk of Bias assessment tool.[6] For each study, several domains were assessed: random sequence generation, allocation concealment, blinding of participants and personnel,

blinding of outcome assessment, completeness of outcome data, selective outcome reporting, and other potential sources of bias. Each domain was categorized as having a low, high, or unclear risk of bias. Detailed risk of bias results for each included study are summarized narratively in the text.

Data synthesis

Data synthesis was conducted using a narrative approach. Where appropriate, a quantitative meta-analysis was performed. Results were presented using summary tables and forest plots. In cases of substantial heterogeneity, a qualitative synthesis was undertaken. Subgroup and sensitivity analyses were planned to explore potential sources of heterogeneity.

Statistical analysis

All analyses were performed using STATA software. Effect estimates for dichotomous outcomes were pooled and expressed as risk ratios (RRs) with corresponding 95% confidence intervals (CIs). Continuous outcomes were synthesized using mean differences (MDs) with 95% CIs.

A DerSimonian–Laird random-effects model was selected a priori to account for anticipated inter-study variability in corticosteroid regimens, patient characteristics, and baseline severity of pneumonia. Between-study heterogeneity was assessed using the I^2 statistic, with values greater than 50% indicating moderate to high heterogeneity. Statistical heterogeneity was outlying evaluated using Cochran's Q test, with $p<0.10$ considered statistically significant. For the primary outcome, robustness of pooled estimates was assessed using leave-one-out sensitivity analysis. Potential publication bias for mortality was evaluated visually using funnel plot symmetry. A two-tailed p value <0.05 was considered statistically significant.

RESULTS

Mortality rate

Twelve studies assessed the mortality rate, with a crude incidence rate of 9.91% (472/4,764) in the intervention group compared to 8.40% (633/7,537) in the control group.

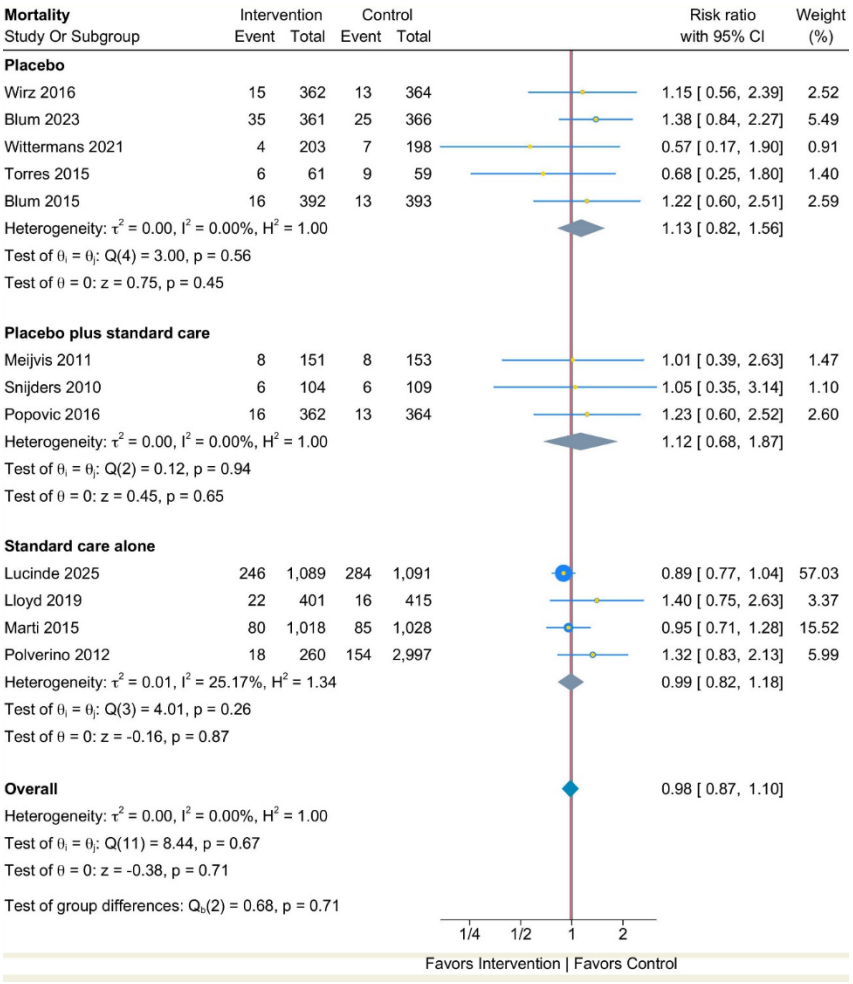

Figure 2: Forest plot comparing the mortality rate between the intervention and control groups, stratified by control type.

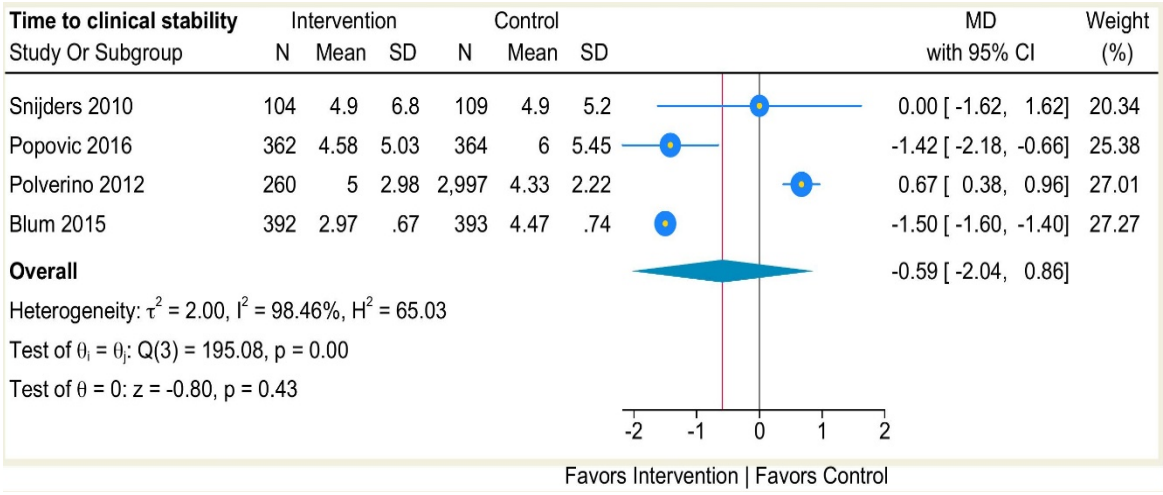

Figure 3: Forest plot comparing time to clinical stability between the intervention and control groups.

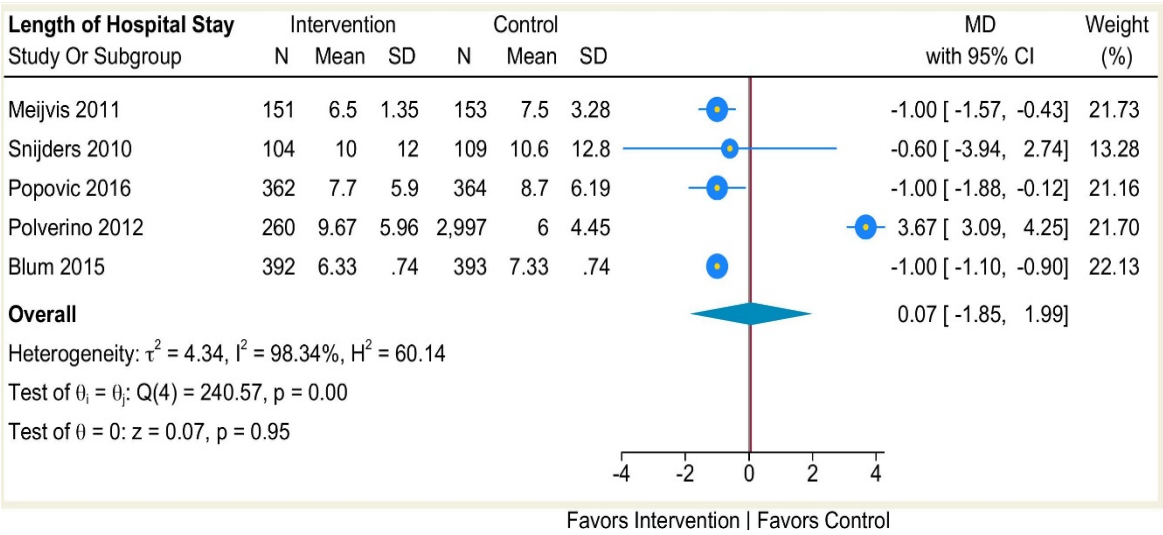

Figure 4: Forest plot comparing length of hospital stay between the intervention and control groups.

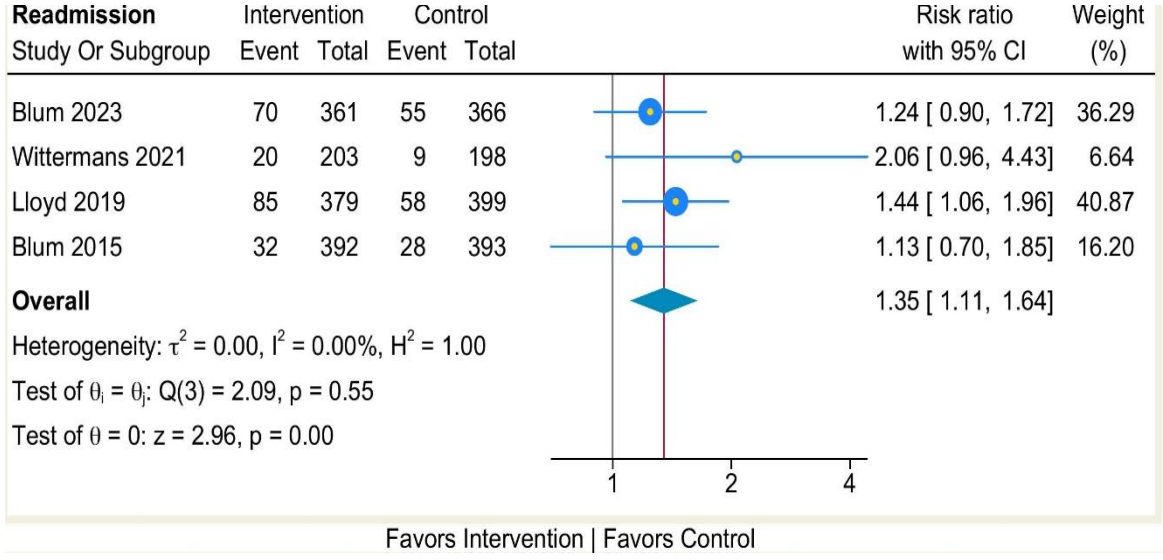

Figure 5: Forest plot comparing the risk of readmission between the intervention and control groups.

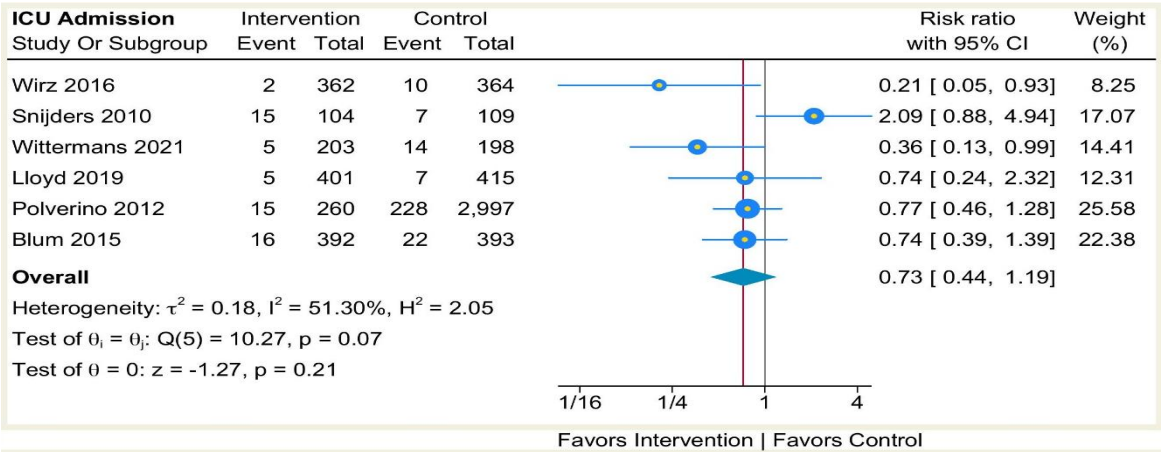

Figure 6: Forest plot comparing the risk of ICU admission between the intervention and control groups.

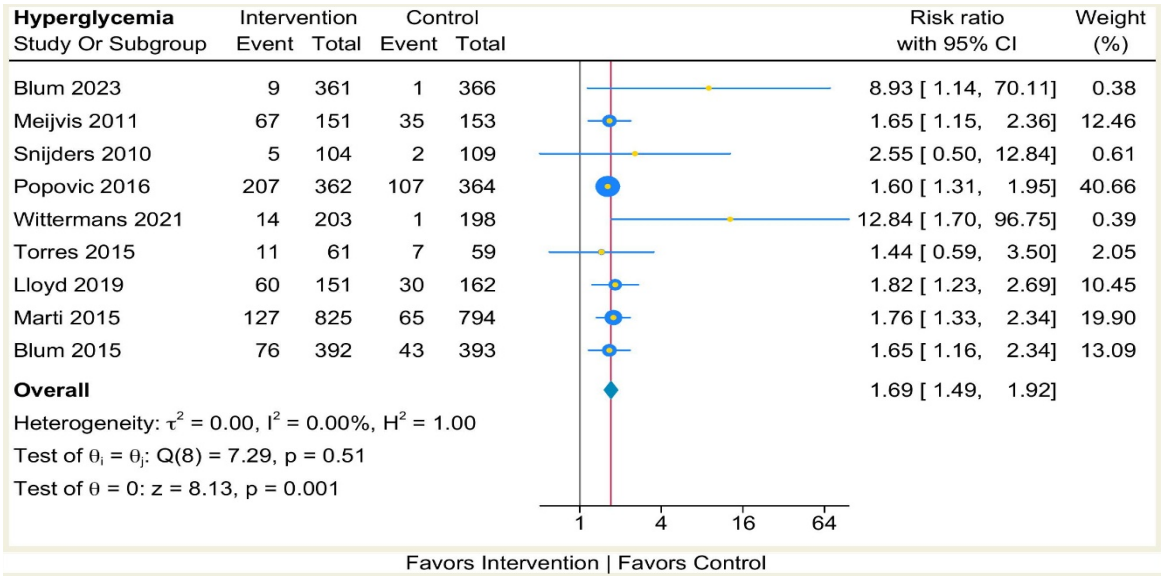

Figure 7: Forest plot comparing the risk of hyperglycemia between the intervention and control groups.

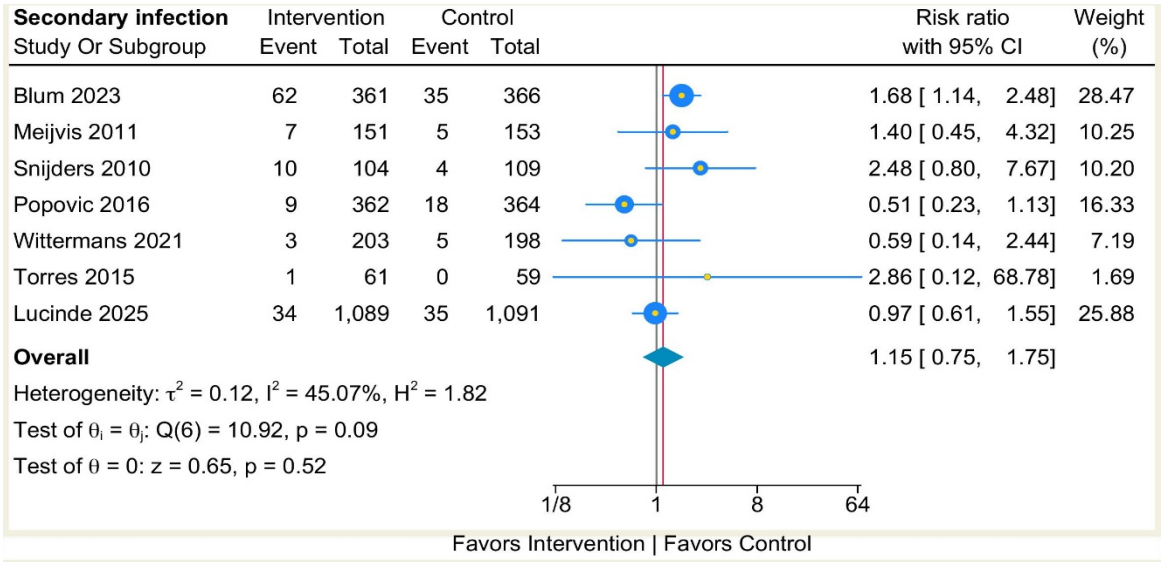

Figure 8: Forest plot comparing the risk of secondary infection between the intervention and control groups.

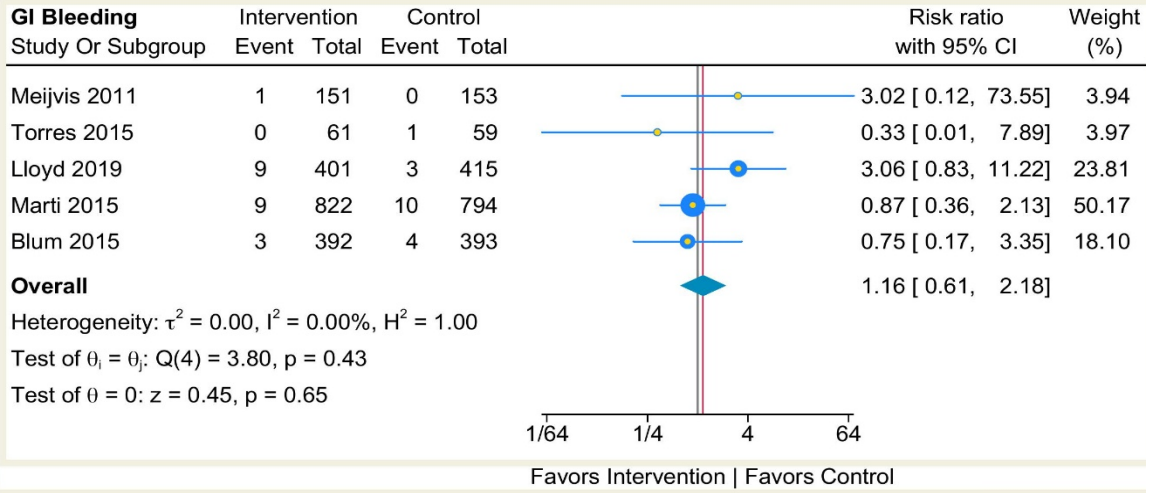

Figure 9: Forest plot comparing the risk of gastrointestinal bleeding between the intervention and control groups.

The pooled analysis showed no statistically significant difference in mortality between the two groups (RR=0.98, 95% CI: 0.87 to 1.10, p=0.71), with no observed heterogeneity (I^2=0.00%, p=0.67). Subgroup analysis by control type revealed no significant difference across the three control subgroups: placebo (RR=1.13, 95% CI: 0.82 to 1.56, p=0.45; I^2=0.00%, p=0.56), placebo plus standard care (RR=1.12, 95% CI: 0.68 to 1.87, p=0.65; I^2=0.00%, p=0.94), and standard care alone (RR=0.99, 95% CI: 0.82 to 1.18, p=0.87; I^2=25.17%, p=0.26) (Figure 2).

Leave-one-out sensitivity analysis confirmed the robustness of the result; no single study materially altered the overall effect estimate.

Clinical recovery outcomes

Pooled analysis of clinical recovery outcomes revealed no significant differences between the intervention and control groups. Specifically, there was no statistically significant reduction in time to clinical stability (MD=−0.59, 95% CI: −2.04 to 0.86, p=0.43; I^2=98.46%, p=0.001; (Figure 3) or length of hospital stay (MD=0.07, 95% CI: −1.85 to 1.99, p=0.95; I^2=98.34%, p=0.001) (Figure 4).

Clinical deterioration outcomes

Regarding clinical deterioration, the pooled analysis demonstrated a statistically significant increase in the risk of readmission in the intervention group compared to the control group (RR=1.35, 95% CI: 1.11 to 1.64, p<0.001; I^2=0%, p=0.55 (Figure 5). In contrast, no significant difference was detected between the two groups regarding the incidence of ICU admission (RR=0.73, 95% CI: 0.44 to 1.19, p=0.21; I^2=51.30%, p=0.07) (Figure 6).

Adverse events

Assessment of adverse events suggested a significantly higher risk of hyperglycemia in the intervention group (RR=1.69, 95% CI: 1.49 to 1.92, p<0.001; I^2=0%, p=0.51 (Figure 7). However, corticosteroid use showed no statistically significant difference in the risks of secondary infection (RR=1.15, 95% CI: 0.75 to 1.75, p=0.52; I^2=45.07% (Figure 8) or gastrointestinal bleeding (RR=1.16, 95% CI: 0.61 to 2.18, p=0.65; I^2=0% (Figure 9).

DISCUSSION

We analyzed twelve randomized controlled trials to assess the impact of early adjunctive corticosteroid therapy on adults with CAP in emergency departments. The accumulated clinical data paint a bleak picture showing corticosteroid use has no survival advantages. It does not accelerate clinical stabilization or hospital discharge. Instead, steroid therapy inflicts a heavier burden of metabolic and post-discharge harm. These results refine existing knowledge and challenge conclusions drawn in earlier trials and meta-analyses.

Our analysis shows absence of mortality benefit; this warrants careful consideration. Dequin et al reported a survival advantage with hydrocortisone use in patients with severe, ICU-level CAP, a finding that generated considerable interest.[7] However, the individual patient data meta-analysis by Briel et al demonstrate that this benefit does not extend to non-severe cases, a distinction that the present analysis reaffirms across a broader and more heterogeneous sample.[8] The observed lack of subgroup difference based on comparator type, along with the near zero heterogeneity in the mortality estimate (I^2=0%), suggests that the likelihood of a genuine effect being hidden by methodological noise is low. The pooled risk ratio of 0.98 is remarkably as close to a null effect as clinical trials are likely to produce.

In Lucinde et al a separate consideration should be given to the mortality data.[9] In this trial conducted in a low-resource setting, a reduction was reported and that appears at odds with our pooled estimate. Varied pathogen ecology, baseline illness severity or background standards

of care might explain this divergence. Therefore, clinicians must exercise caution when applying findings from low-resource pragmatic trials to higher-income emergency department populations. By contrast, Lloyd et al and Wittermans et al found no survival benefit particularly pronounced outside of severe or ICU settings.[10,11] These findings reinforce the null conclusion.

Clinical recovery demands equally careful evaluation. Earlier investigators like Blum et al and Siemieniuk et al reported findings attracting widespread attention with shorter time to clinical stability and reduced hospital duration.[12,13] But, replication of those findings has proven inconsistent. Popovic et al and Lloyd et al demonstrated little or no benefit in comparable patient cohorts.[10,14] Our analysis, while directionally neutral, was accompanied by extraordinary heterogeneity (I^2 exceeding 98%) in both recovery outcomes. Such heterogeneity represents more than statistical noise; it signals extreme sensitivity to contextual factors. Which cannot be adequately controlled in standard meta-analytic pooling. So clinicians must hesitate before applying trial-level observations to individual patients.

Perhaps the most clinically significant finding in our analysis is the elevated risk of hospital readmission among those who received corticosteroids. This outcome received insufficient attention in prior reviews. One plausible explanation is the anti-inflammatory properties of corticosteroids in the long term. By suppressing CRP and other inflammatory markers as documented by Snijders et al leading to apparent clinical stabilization and discharge without achieving true resolution of infection.[15] The result is a patient who meets discharge criteria sooner but remains vulnerable to relapse once the drug effect wanes, a pattern corroborated by the long-term follow-up data from Blum et al suggesting a late signal of infectious complications.[16] The readmission finding therefore cannot be dismissed as a statistical curiosity; it has direct implications for how corticosteroid-treated patients are monitored after discharge.

Another relevant outcome is the failure to reduce the ICU admission rate. This reinforces the impression that corticosteroids, at the doses and timing studied, are unable to halt meaningful clinical deterioration. The one clearly consistent adverse signal is hyperglycemia. This outcome is unsurprising and mechanistically predictable, but its magnitude (RR=1.69) underscores that this is not a trivial or manageable side effect, especially in a population that harbors diabetes, impaired glucose regulation, or steroid sensitivity. Torres et al and Wirz et al, advocate for a biomarker-stratified approach.[17,18] This strategy reserves corticosteroid use for patients with evidence of hyperinflammation such as CRP level exceeding 150 mg/l. The concept remains biologically compelling, but prospective evaluation must validate this protocol to enrich the target population toward those most likely to benefit and protect low-risk patients from harm.

Our methodology improves on prior syntheses. We incorporated recent trial data, applied leave-one-out sensitivity testing, and evaluated a wider spectrum of outcomes. Crucially, this included hospital readmissions. Earlier reviews ignored this endpoint. The robustness of the primary mortality estimate across sensitivity analyses offers reasonable confidence in the null conclusion for that outcome.

Limitations

Several limitations merit acknowledgement. The extraordinary heterogeneity observed in clinical recovery outcomes (I^2>98%) signals genuine variability in treatment effects across contexts driven by differences in corticosteroid regimens, patient case-mix, timing of initiation, and how clinical stability was defined and measured and cautions against treating pooled recovery estimates as point estimates applicable to any individual clinical scenario. Analysis at the study rather than patient level precluded exploration of effect modification by individual-level factors such as disease severity, comorbidity burden, or baseline inflammatory status characteristics that are likely to be the key determinants of who, if anyone, stands to benefit. Finally, the majority of included trials had relatively short follow-up horizons, meaning that delayed harms most notably post-discharge infectious complications may be underrepresented in the available evidence. Future work should place individual patient data meta-analysis and trials with extended post-hospitalization surveillance at the centre of the research agenda.

CONCLUSION

In adults presenting to the emergency department with community-acquired pneumonia and who do not require immediate intensive care, early administration of systemic corticosteroid therapy has not demonstrated a significant clinical benefit, since current evidence indicates no significant reduction in mortality, time to clinical stability, or length of hospital stay. In contrast, it has been consistently associated with elevated risk of hyperglycemia and hospital readmission, two outcomes with direct important implications on patient welfare and healthcare resource utilization. Accordingly, the routine corticosteroid use in non-ICU CAP patients is not considered supported by the available evidence studied. A more targeted strategy, guided by markers of inflammatory phenotype and reserved for patients with a demonstrable hyperinflammatory response, is both biologically rational and carry a priority for future large-scale trials. Until more definitive evidence becomes thoroughly investigated, clinicians should weigh the modest and uncertain clinical gains against the reliably observed adverse effects before initiating corticosteroids in the non-ICU CAP patient.

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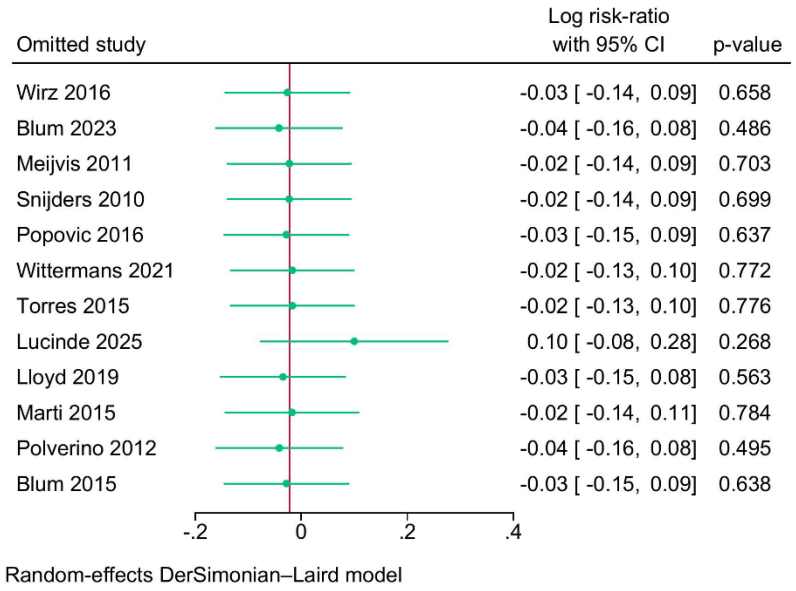
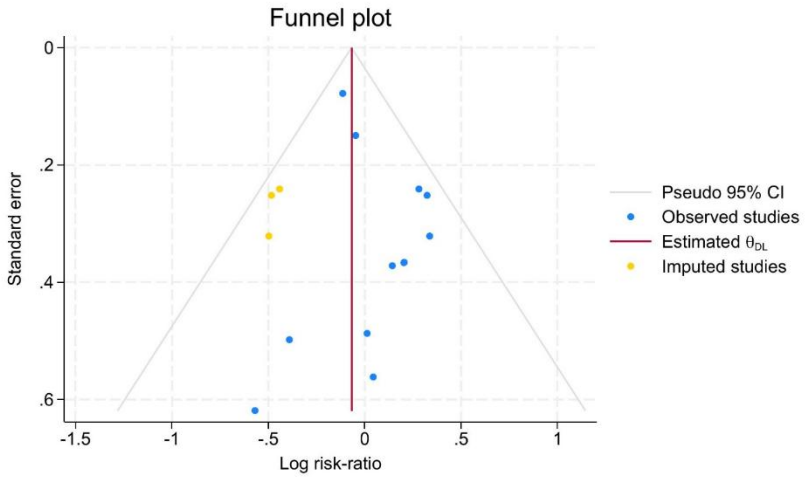
ANNEXURE



Supplementary Figure 1



Supplementary Figure 2